The Editors of the Proceedings of the Nutrition Society *accept no responsibility for the abstracts of papers read at the Society's meetings for original communications.*

PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

A meeting of the Nutrition Society was held in the Boole II Lecture Theatre, University College, Cork, Republic of Ireland on Wednesday and Thursday, 13/14 September 1989, when the following papers were read:

https://doi.org/10.1079/PNS19900014 Published online by Cambridge University Press

Blood carotenoid profiles in Irish population groups. By VALERIE O'DONNELL, K. R. O'SULLIVAN and P. M. MATHIAS, *Department of Biological Sciences, Dublin Institute of Technology, Kevin Street, Dublin 8, Republic of Ireland*

The current interest in the possible role of carotenoids against cancer has generated numerous studies on blood carotenoid profiles in humans (Bieri *et al.* 1985, Thurnham *et al.* 1988). In the present study, baseline concentrations in plasma of total carotenoids (CAR), two non-provitamin A carotenoids, lutein (LUT) and lycopene (LYC), and three provitamin A carotenoids, β-cryptoxanthin (BCP), α-carotene (AC) and β-carotene (BC), were estimated in healthy Irish males (*n* 14) and females (*n* 22), aged 19–22 years, using high performance liquid chromatography (HPLC). Results are shown in the Table.

		Carotenoids (μg/l)											
		CAR		LUT		BCP		LYC		AC		BC	
Sex	*n*	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Males	14	889*	394	62	26	69*	56	56	34	37	27	154	127
Range		200–1440		20–102		55–255		18–132		19–89		50–403	
Females	22	1202	220	77	38	278	280	64	35	39	22	181	92
Range		550–2800		42–174		49–520		09–74		14–89		120–480	

*Statistically significantly different from females (Mann-Whitney U test): $P<0{\cdot}01$.

Values for total and individual carotenoids were similar to those reported by other workers (Willet *et al.* 1983; Thurnham & Flora, 1988) with BC accounting for approximately 15% of total carotenoids. CAR and BCP were significantly lower in males, in agreement with the findings of Thurnham & Flora (1988) who, in a much larger population group, reported significantly lower levels of most carotenoids in males compared with females. In our study no significant difference was found in daily carotene intake between males (8·0 (SE 5·2) mg) and females (6·2 (SE 3·4) mg) when estimated by food-frequency questionnaire. The observed sex differences in plasma carotenoids could be explained in part by a higher cigarette and alcohol consumption in males. These have been shown to have a depressive effect on plasma carotenoids (Scott-Stryker *et al.* 1988).

In a sample of older subjects (mean age 63 years) with colorectal cancer (*n* 9), total carotenoids (578 (SE 127) μg/l) and lutein (61 (SE 8) μg/l) were found to be significantly lower ($P<0{\cdot}01$ and $P<0{\cdot}001$ respectively) than those levels present in an age- and sex-matched control group (*n* 9, 863 (SE 201) and 94 (SE 14) μg/l respectively). Carotenoid intakes were lower and alcohol consumption higher in the cancer group (O'Sullivan *et al.* 1990).

Bieri, J. G., Brown, E. D. & Smith, J. C. (1985). *Journal of Liquid Chromatography* **8**, 473–484.
O'Sullivan, K. R., Mathias, P. M., Tobin, A. & O'Morain, C. (1990). *Proceedings of the Nutrition Society* **49**, 91A.
Scott-Stryker, N., Kaplan, L. A., Stein, E. A., Stampfer, M. J., Sober, A. & Willet, W. C. (1988). *American Journal of Epidemiology* **127**, 283–296.
Thurnham, D. I. & Flora, P. S. (1988). *Proceedings of the Nutrition Society* **47**, 181A.
Thurnham, D. I., Smith, E. & Flora, P. S. (1988). *Clinical Chemistry* **34**, 377–381.
Willet, W. C., Stampfer, M. J., Underwood, B. A., Taylor, J. O. & Hennekens, C. H. (1983). *American Journal of Clinical Nutrition* **38**, 559–566.

Comparison of computerized nutrient calculations and analysed values for water, protein, fat, calcium, iron, sodium and potassium in cooked dishes eaten in Ireland. By EAVAN SALMON and P. M. MATHIAS, *Department of Biological Sciences, Dublin Institute of Technology, Kevin Street, Dublin 8, Republic of Ireland*

Ireland has its own typical foods and composite dishes for which there is little nutritional composition information to date. The use of computer software to calculate estimates of the nutrient composition of foods and recipes as eaten can provide a rapid and cheap method for the analysis of many dishes. To assess the validity and relative accuracy of this method the present study compared the values obtained for moisture, protein, fat, calcium, iron, sodium and potassium in seventeen mixed dishes by computerized nutrient calculation with those values obtained from standard methods of laboratory analysis following preparation and cooking of these dishes. The 'Yield Factor Method' (Powers & Hoover, 1989) was used for computerized recipe calculation. The information collected was coded according to tables of food composition (Paul & Southgate, 1978) and nutrient composition computed using the 'Microdiet' program of these same tables. The results for laboratory (L) and computerized (C) analysis are shown in the Table.

	Moisture (%)		Protein (g)		Fat (g)		Ca (mg)		Fe (mg)		Na (mg)		K (mg)	
Dish	L	C	L	C	L	C	L	C	L	C	L	C	L	C
Coddle	70	84	6·0	3·6	4·9	5·2	16	12	0·50	0·43	389	212	189	131
Irish stew	78	73	7·9	4·8	6·4	5·2	16	11	0·55	0·55	61	72	344	155
Colcannon	82	81	2·5	1·7	6·4	7·6	48	37	0·34	0·28	70	100	209	209
Champ	78	80	2·5	1·7	4·6	5·6	32	29	0·35	0·25	112	79	234	269
Pork ciste	67	64	10·5	11·5	12·9	14·9	79	64	1·42	1·40	179	158	239	148
Chowder	73	73	7·6	5·6	4·9	5·0	102	73	0·42	0·42	125	99	275	227
Potato cake	48	54	2·6	2·3	20·4	22·0	70	60	0·53	0·60	258	251	385	215
Potato soup	82	73	4·3	3·9	8·5	10·9	65	48	0·31	0·30	177	225	240	245
Lasagne	63	67	11·4	8·7	11·1	10·9	137	101	1·05	1·20	248	174	326	228
Cottage pie	68	79	8·1	7·7	7·8	8·5	28	23	1·10	1·30	153	195	369	150
Chicken pie	70	56	12·2	15·5	6·9	7·6	22	12	0·94	0·90	366	318	225	215
Fish pie	75	68	9·9	6·7	6·2	5·9	65	50	0·37	0·34	68	154	256	243
Lamb curry	69	63	13·6	10·4	16·5	16·1	33	30	1·31	2·30	125	143	141	192
Chicken curry	75	68	15·8	12·5	8·9	9·9	35	29	0·88	1·80	133	80	262	262
Beef curry	70	65	17·7	12·3	12·2	11·5	28	29	1·25	2·40	92	184	125	193
Beef goulash	72	76	8·7	6·5	5·0	5·7	22	15	0·90	0·87	101	94	412	233
Bolognese meat sauce	79	75	10·4	8·9	8·8	9·4	32	38	1·37	1·70	161	126	366	315

Overall there was good agreement between the two methods of analysis for all nutrients tested, although there was a tendency for the underestimation of protein by computer calculation. This was probably due to inaccuracies in yield factor determination, especially for meat, where cooking time and temperature, the shape and size of the meat itself, and the proportion of fat and bone can affect yield factors. Variations between laboratory and computerized analysis for Na and K in some dishes could be explained, in part, by natural variations of food content of these nutrients and the effects of washing and preparation of ingredients before cooking. Nevertheless this study indicates that computer calculation of recipes is generally reliable.

Paul, A. A. & Southgate, D. A. T. (1978). *McCance and Widdowson's The Composition of Foods*. London: H.M. Stationery Office.

Powers, P. M. & Hoover, L. W. (1989). *Journal of the American Dietetic Association* **89**, 224–233.

The association between diet and colorectal polyps and cancer in an Irish population. By KATHRYN R. O'SULLIVAN and P. M. MATHIAS, *Department of Biological Sciences, Dublin Institute of Technology, Kevin Street, Dublin 8* and A. TOBIN and C. O'MORAIN, *Trinity College, Dublin 2, Republic of Ireland*

In a previous communication (O'Sullivan *et al.* 1989), a significant association was found between low blood levels of antioxidants (such as vitamins E and A, and selenium) in patients with colonic polyps and colorectal cancer. To assess the relationship between diet and these nutrient levels, control (*n* 23), polyp (*n* 18) and cancer (*n* 16) subjects were randomly selected from the main study population and two types of dietary histories taken: (1) a detailed 3-d dietary recall to assess current intakes and hence possible influence on blood nutrients; (2) a food-frequency questionnaire to assess the possible long-term (i.e. past 20 years) relationship not only between diet and biochemical status but also between diet and the genesis of cancer. No significant differences were found between the three groups in either the macro- or micronutrient intakes in both the recall and questionnaire. The mean body mass index (BMI) was similar in each group. Nutrient values were categorized into tertiles based on their distribution in controls and odds ratios (OR) of cancer and polyp risk by tertiles calculated using the Mantel–Haenzel Logit model. Results are shown in the Table along with OR and 95% confidence intervals (CI) of cancer and polyps risk among controls.

Nutrient	Highest tertile	OR	95% CI
Energy (MJ/d)	>11·0	2·03	0·7– 6·1
Protein (g/d)	>85·7	2·1	0·7– 6·2
Fat (g/d)	>116	2·7	0·9– 8·0
Alcohol (g/d)	>14·6	2·2	0·7– 7·4
%Fat (as energy)	>40·5	0·5	0·2– 1·4
%Alcohol (as energy)	>2·8	1·7	0·6– 5·0
Carotene (μg/d)	<4496	2·13	0·7– 6·6
Breakfast cereals	<3 times/week	2·5	0·8– 7·9
Fruit	<7 portions/week	3·3	1·0–10·0
Carrots	<once/week	4·7	0·5–42·0
Butter	>300 g/week	3·6	1·2–11
Cheese	>125 g/week	1·97	0·7– 5·8

The greatest association for increased risk of cancer was found for higher intakes of total fat and butter, and for lower intakes of fruit and, perhaps, carrots. This is in general agreement with the studies of Graham (1983) and Klatsky *et al.* (1988).

In conclusion, the present study suggests that differences in blood antioxidant status in the study groups are not associated with diet and that other dietary factors, such as high-fat and low-fibre intakes may be more relevant to cancer risk.

This work is funded by the Irish Cancer Society.

Graham, S. (1983). *Cancer Research* **43**, 2409–2413.

Klatsky, A. L., Armstrong, M. A., Friedman, G. D. & Hiatt, R. A. (1988). *American Journal of Epidemiology* **128**, 1007–1015.

O'Sullivan, K. R., Mathias, P. M., Tobin, A. & O'Morain, C. (1989). *Proceedings of the Nutrition Society* **48**, 135A.

Iron deficiency anaemia in long-term institutionalized elderly women in Northern Ireland.

By JILL EATON-EVANS[1], LINDA WALKER[2], T. R. O. BERINGER[2], D. H. GILMORE[2] and J. J. STRAIN[1], [1]*Biomedical Sciences Research Centre, University of Ulster at Jordanstown, Newtownabbey, Co. Antrim BT37 0QB* and [2]*Geriatric Medical Unit, Royal Victoria Hospital, Grosvenor Road, Belfast BT12 6BA, Northern Ireland*

Although anaemia is a common occurrence in elderly populations, including the institutionalized elderly, there is much debate on its aetiology (Timiras & Brownstein, 1987). Iron deficiency is considered to be an important contributor to anaemia in the hospitalized elderly, yet there is accumulating evidence that, in general, Fe-deficiency anaemia is rare among the elderly in western societies (Dallman *et al.* 1984). In the present study, blood samples from elderly women were analysed for Fe deficiency and Fe deficiency anaemias and compared with similar values from younger, post-menopausal women.

Dietary, biochemical, anthropometric and medical data were collected from long-term, medically stable, institutionalized elderly women (*n* 20) in a geriatric medical unit (GMU) in Northern Ireland. From observed household measures of food consumed over 2 d, dietary Fe intake was estimated at 7·2 (SD 2·8) mg/d. Blood samples were analysed at the Royal Victoria Hospital laboratories by standard methods and compared (*t* test) with similar measurements taken from a random sample of post-menopausal (PM) women (age range 50–64 years) in the Northern Ireland population (Barker *et al.* 1989). Fe stores were calculated using the equations of Ballot *et al.* (1989). Mean values of the blood measurements are given in the Table.

Subjects		*n*	Age (years)	Hb (g/l)	SF (μg/l)	TS (%)	MCHC (%)	Fe Stores (mg)	MCV (fl)
GMU	Mean	20	85	127*	139*†	21†	33·9	10·8†	87·0
	SEM		17	3·1	58·2	2·4	1·1	1·6	3·2
PM	Mean	62	57	135	68	19‡	33·1	9·6	87·9
	SEM		0·5	1·7	8·3	0·8	0·5	0·8	1·5

Significantly different from PM women: *$P<0·05$.
†*n* 18, ‡*n* 55.

Haemoglobin (Hb) was significantly decreased while serum ferritin (SF) was significantly increased in the elderly (GMU) women compared with the younger (PM) women. Transferrin saturation (TS) together with mean corpuscular haemoglobin concentration (MCHC), mean cell volume (MCV) and Fe stores did not differ. These results suggest that the Fe status measurements in the elderly women reflect the anaemia of chronic disease. Six of these elderly women were classified as anaemic (Hb<120 g/l) but, using the criteria of Ballot *et al.* (1989), only one was classified as Fe deficient.

Ballot, D. E., MacPhail, A. P., Bothwell, T. H., Gillooly, M. & Mayet, F. G. (1989). *American Journal of Clinical Nutrition* **49**, 156–161.

Barker, M. E., McClean, S. J., McKenna, P. G., Reid, N. G., Strain, J. J., Thompson, K. A., Williamson, A. P. & Wright, M. E. (1989). In *Diet, Lifestyle and Health in Northern Ireland*. Coleraine: University of Ulster.

Dallman, P. R., Yip, R. & Johnson, C. (1984). *American Journal of Clinical Nutrition* **39**, 437–445.

Timiras, M.-L. & Brownstein, H. (1987). *Journal of American Geriatric Society* **35**, 639–643.

Nutrient intakes by vegetarians in Newcastle upon Tyne. By BEATRICE T. ROBERTS and J. C. MATHERS, *Department of Agricultural Biochemistry and Nutrition, The University, Newcastle upon Tyne NE1 7RU*

Vegetarians are a significant minority within the UK whose dietary choices may have health consequences.

Thirty-one vegetarians (twenty-four females and seven males, aged 19–48 years) were recruited during late 1987 in the Newcastle area by advertising in the university, through a local radio programme and by personal contact. With the exception of one woman who occasionally ate fish, none of the subjects ate animal flesh. Eighteen of the volunteers were students and most (18/31) had been vegetarians for 1–5 years with only three (all women) being vegetarians for more than 10 years. None had been brought up as a vegetarian and most (25/31) gave moral considerations as their main reason for choosing a vegetarian diet. Four men and eight women were smokers. Food intake was measured by the 7-d weighed inventory method and energy and nutrient intakes calculated using UK food tables with the aid of the 'Microdiet' package (Bassham & Fletcher, 1985).

				Daily intakes of energy and selected nutrients						
		Height (m)	Quetelet index	Energy (MJ)	Protein (g)	Fat (g)	Dietary fibre	Calcium (mg)	Iron (mg)	Vitamin C (mg)
Female	Mean	1·67	22	8·1	58	72	32	842	16	114
(*n* 24)	SEM	0·010	0·5	0·88	6·3	12·7	6·6	126·2	2·6	28·1
Male	Mean	1·78	23	12·3	85	100	40	1230	20	140
(*n* 7)	SEM	0·023	0·6	2·30	19·6	22·1	16·1	384·0	7·1	64·7

Our subjects were taller and lighter than the UK adult means (Knight, 1984), possibly reflecting their predominantly non-manual socioeconomic backgrounds. With the exceptions of zinc (9 mg/d) and vitamin B_{12} (1·7 μg/d), where intakes were possibly marginal, energy and nutrient intakes were adequate by conventional standards. Daily sodium (2·6 g) and chloride (3·8 g) intakes were similar to those of other Newcastle subjects (Mathers, 1988). The percentages of energy obtained from fat, protein, carbohydrate and alcohol were 33, 12, 50 and 6, and 30, 12, 51 and 7 for women and men respectively, and were in line with recommendations made by the COMA report (Department of Health and Social Security, 1984).

We thank the Vegetarian Society UK for assistance with this project.

Bassham, S. & Fletcher, L. R. (1985). *Proceedings of the Nutrition Society* **44**, 36A.

Department of Health and Social Security (1984). *Diet and Cardiovascular Disease. Committee on Medical Aspects of Food Policy. Report on Health and Social Subjects* no. 28. London: H.M. Stationery Office.

Knight, I. (1984). *The Heights and Weights of Adults in Great Britain*, Office of Population Censuses and Surveys. London: H.M. Stationery Office.

Mathers, J. C. (1988). *Journal of Human Nutrition and Dietetics* **1**, 155–161.

An evaluation of a high-fibre diet in the dietetic treatment of patients on regular haemodialysis. By JUDITH M. MCDONALD, *Department of Nutrition and Dietetics, Belfast City Hospital, Belfast BT9 7AB* and J. J. STRAIN, *Biomedical Sciences Research Centre, University of Ulster at Jordanstown, Newtownabbey, Co. Antrim BT37 0QB, Northern Ireland*

The major objectives of the dietary management of patients with end-stage renal disease are to maintain nitrogen, energy and water balance and acceptable serum ranges of urea, potassium and phosphate. This has traditionally been achieved by highly refined diets. Patients with chronic renal disease, however, have a high incidence of atherosclerotic complications and it may be prudent to attempt to increase dietary fibre intakes of these patients. Recent work suggests that increased dietary fibre intakes may not significantly increase dietary or serum K and phosphate (McKenzie & Henderson, 1986) and indeed may reduce plasma urea levels (Rampton *et al.* 1984). The aim of the present investigation was to evaluate the use of a high-fibre (HF) diet by patients receiving twice weekly, 5–8 h sessions of haemodialysis.

A total of twelve patients (six men, six women), mean age 52 (range 28–69) years, who were following a standard 60 g protein/d, low-fibre (LF) diet, participated in the study. After an initial 2 month 'run in' period (phase 1) the patients were instructed to follow a 60 g protein/d, HF diet for 2 months (phase 2) and then asked to revert to the LF diet for a further 2 months (phase 3). Compliance on diets in phases 1 and 2 was monitored by 7-d weighed dietary records, and reported dietary fibre intakes (g/d) were 10·1 (SD 1·35) and 16·8 (SD 2·29) for the LF and HF diets respectively. Mean blood biochemistry values from monthly samples during the three phases were compared by analysis of variance.

	K (mM)	Urea (mM)	Phosphate (mM)	TG (mM)	TC (mM)	HDL-C (mM)
Phase 1	5·62[a]	27·23[a]	2·15[a]	2·08[a]	6·17[a]	0·98[a]
Phase 2	6·01[a]	30·05[b]	2·29[a]	2·01[a]	6·58[b]	1·10[b]
Phase 3	5·65[a]	27·81[a]	2·04[a]	ND	ND	ND
Treatment SE	0·185	1·049	0·132	0·127	0·160	0·038

TG, triacylglycerols; TC, total cholesterol; HDL-C, high density lipoprotein-cholesterol; ND, not determined.

[a],[b] Means in vertical columns with different superscript letters are significantly different (least significant test): $P<0·05$.

The significant increase in serum urea and the large increases by some patients in serum K would suggest that dietary fibre intake by twice weekly haemodialysis patients should not be increased above the traditionally low levels without careful monitoring of blood biochemistry.

McKenzie, S. I. & Henderson, I. S. (1986). In *Aspects of Renal Care*, vol. 1, pp. 172–177 [E. Stevens and P. M. Monkhouse, editors]. London: Bailliere Tindall.

Rampton, D. S., Cohen, S. L., Crammond, V. de B., Gibbons, J., Lilburn, M. F., Rabet, J. Y., Vince, A. J., Wager, J. D. & Wrong, O. M. (1984). *Clinical Nephrology* **21**, 159–163.

Dietary supplementation in undernourished children with cystic fibrosis. By ALICIA PIERCE[1], A. FLYNN[1], J. B. G. WATSON[2] and P. A. MORRISSEY[1], [1]*Department of Nutrition, University College, Cork* and [2]*Department of Paediatrics, Regional Hospital, Cork, Republic of Ireland*

Malnutrition, as evidenced by a failure to achieve normal growth curves, often accompanies the poor respiratory status seen in children and adolescents with cystic fibrosis (CF). A close, inverse correlation between the degree of underweight and survival has been demonstrated (Kraemer *et al.* 1978). While the relationship between nutritional and pulmonary status is complex, success in improving the clinical course of CF has been demonstrated using nutritional rehabilitation techniques (Levy *et al.* 1985).

In the present study, seven CF patients (age range 6·3–20 years) with body-weights below the 10th percentile for age and sex received nocturnal nasogastric supplementation over a fourteen-night period. The supplementation product contained, per litre, 45 g protein (as free amino acids and oligopeptides), 16·7 g fat (52% as medium-chain triacylglycerols), 167·5 g carbohydrate (as maltodextrin), 4·2 MJ energy, and various vitamins and minerals. The supplementation volume, calculated from each patient's age and sex, provided an extra 20–50% of the recommended daily amount (RDA) (Department of Health and Social Security, 1979) for energy in addition to *ad lib.* oral intake. Daily energy and nutrient intakes of patients were calculated from 3-d weighed and measured records of all foods, beverages and vitamins taken in the week before supplementation and in the week after supplementation.

Effect of nocturnal nasogastric supplementation on body anthropometric variables and food intake

		Before supplementation			After supplementation			Significance of difference‡:
	n	Mean	SD	Range	Mean	SD	Range	*P*=
Wt (kg)	7	31·9	13·5	13·3–54·4	34·2	14·5	15·1–58·9	0·0018
TSF (mm)	7	4·7	1·5	2–6	5·3	2·2	2–8	0·17
MAC (mm)	7	181	40	120–240	189	42	125–251	0·0007
Energy intake*	5	134·2	28·6	108–173	167·0	88·1	89–308	0·39
Protein intake†	5	14·0	4·9	8·7–21·4	18·4	2·8	15·6–22·4	0·028
Fat intake†	5	31·3	7·8	20·2–41·2	34·3	9·9	22·3–46·1	0·45
Carbohydrate intake†	5	54·5	8·5	46·0–46·3	47·4	11·0	31·6–59·0	0·052

TSF, triceps skin-fold thickness; MAC, mid-arm circumference.
*As % RDA for age and sex.
†As % total energy.
‡Paired *t* test.

The results show that body-weight (P=0·0018) and mid-arm circumference (P=0·0007) increased significantly after supplementation. Protein energy also increased significantly (P=0·028), while changes in energy, fat and carbohydrate were not significant. It does appear that nocturnal nutritional supplementation via nasogastric tube, while allowing *ad lib.* oral intake during the day, may be an effective means of achieving short-term improvements in growth status. The wide range of energy intakes suggest that some of our CF patients may experience difficulty in achieving the recommended intakes of 120–150% of RDA for energy in order to compensate for losses due to malabsorption and increased metabolic demand due to respiratory pathology.

Department of Health and Social Security (1979). Recommended daily amounts of food energy and nutrients for groups of people in the United Kingdom. *Report on Health and Social Subjects* no. 15. London: H.M. Stationery Office.

Kraemer, R., Rudberg, A., Hadron, B. & Rossi, E. (1978). *Acta Paediatrica Scandinavica* **67**, 33–37.

Levy, L. D., Durie, P. R., Pencharz, P. B. & Corey, M. L. (1985). *Journal of Pediatrics* **107**, 225–230.

Macronutrient intake and digestibility in eighteen cystic fibrosis patients. By A. O'RAWE, J. THOMPSON and J. A. DODGE, *Department of Child Health, Queen's University of Belfast, Institute of Clinical Science, Royal Victoria Hospital, Belfast* and A. O. B. REDMOND, *Royal Belfast Hospital for Sick Children, Belfast* and K. J. MCCRACKEN, *Food and Agricultural Research Division, Department of Agriculture Northern Ireland* and *The Queen's University of Belfast, Newforge Lane, Belfast BT9 5PX, Northern Ireland*

The body mass deficit in cystic fibrosis (CF) is a major factor adversely affecting prognosis (Kraemer *et al.* 1978). Median age of survival between two centres may vary by as much as 9 years with differences in nutritional management being the only difference in patient care (Corey *et al.* 1988).

We reviewed existing dietary practices in our clinic in eighteen CF patients (mean age 14 (SE 1·3) years) to assess the success of our policy of an energy-rich, non-restricted-fat diet (energy 130–150% recommended daily amount (RDA); Department of Health and Social Security, 1979).

Analyses of nutrient and energy intakes were made by 7-d weighed food intakes. Faecal collections were made for 3 d using radio-opaque pellets as markers to determine nutrient digestibility in eleven subjects. The mean body mass index of the whole group was 95·4 (SE 2·9)% of normal weight for height. The mean energy intake was 100·2 (SE 4·2)% of RDA, with fat contributing 36% and protein 13·2%. Energy intake ranged from 72 to 141% RDA and there was a tendency for fat intake to be lower for girls (93·4 g/d, *n* 8) compared with boys (105·6 g/d, *n* 10). The digestibility coefficients for dry matter, fat, protein and energy were close to normal values for seven of the eleven subjects, being 0·94, 0·95, 0·87 and 0·93 respectively. The other four subjects, all of whom were receiving pancreatic enzyme supplementation, had values which were significantly below the normal range (Table). In the case of patient A, the low energy digestibility resulted in an effective energy intake which was only 60% of RDA. This individual had a body mass index of 86·2% of normal weight for height.

Digestibility coefficients for dry matter, fat, protein and energy

Patient	Dry Matter	Fat	Protein	Energy
A	0·76	0·65	0·51	0·67
B	0·91	0·90	0·85	0·88
C	0·85	0·77	0·75	0·80
D	0·87	0·81	0·81	0·83

These results demonstrate that the advice given to encourage high-energy consumption in CF patients has not been effective. In addition a small number of patients may be further compromised due to high faecal energy losses.

This study emphasizes the need to examine the energy balance equation on an individual patient basis and relate it to anthropometric measurements, metabolic rate and pulmonary function score.

Corey, M., McLaughlin, F. J., Williams, M. & Levison, H. (1988). *Journal of Clinical Epidemiology* **41**, 588–591.

Department of Health and Social Security (1979). Recommended daily amounts of food energy and nutrients for groups of people in the United Kingdom. *Report on Health and Social Subjects* no. 15. London: H.M. Stationery Office.

Kraemer, R., Rudberg, A., Hadorn, B. & Rossie, E. (1978). *Acta Paediatrica Scandinavica* **67**, 33–37.

Dietary energy intake and faecal energy excretion in normal healthy children. By JANE MURPHY, SARAH BOND and S. A. WOOTTON, *Department of Human Nutrition, Southampton University, Southampton SO9 3TU*

There is relatively little information on dietary energy intakes and faecal energy excretion in normal, healthy children. Moreover, the origins of energy within the stool are uncertain. The purpose of the present study was to (1) compare the gross energy intake with the metabolizable energy intake estimated from food composition tables, and (2) describe the energy, lipid and protein contents of the stool in normal, healthy children.

Nineteen healthy children (eight male, eleven female), mean age 10 (SE 0·4) years, range 8–14 years, recorded weighed food intakes for 7 d and collected stools for the final 3 d between carmine markers. Energy intake was estimated using computerized food composition databases and expressed as (1) gross energy intake, using heats of combustion values (Merrill & Watt, 1955) for protein (23·6 kJ/g), lipid (39·3 kJ/g), carbohydrate (17·4 kJ/g) and fibre (17·4 kJ/g), and (2) metabolizable energy intake, using modified Atwater factors for protein (17 kJ/g), lipid (37 kJ/g) and carbohydrate (16 kJ/g) (Paul & Southgate, 1978). The weighed stools were analysed for energy (bomb calorimetry), nitrogen (Kjeldahl) and lipid (Van de Kamer *et al.* 1949) in order to estimate faecal energy and the energy within the stool provided by lipid and protein (N×6·25) using the relevant heat of combustion factors. The results are summarized in the Table.

	GEI (kJ/d)	MEI (kJ/d)	GEI–MEI (kJ/d)	FE (kJ/d)	FL (kJ/d)	FP (kJ/d)
Mean	9289	7954	1336	327	83	167
SE	255	209	54	33	6	17
Minimum	7437	6397	1040	123	43	63
Maximum	11 191	9453	1740	647	140	348

GEI, gross energy intake; MEI, metabolizable energy intake; FE, faecal energy; FL, faecal lipid; FP, faecal protein.

Gross energy intake was approximately 17% greater (range 14–20%) than metabolizable energy intake estimated using modified Atwater factors. The difference in gross energy intake calculated with and without consideration of dietary fibre intake was approximately 4% (range 2–6%). Faecal energy was approximately 3·5% (range 1·3–5·8%) of gross energy intake. There appeared to be some relationship between faecal protein and faecal energy (r 0·789), faecal protein accounting for approximately 54% of the energy within the stool. A weaker relationship between faecal lipid and faecal energy was observed (r 0·470), with faecal lipid accounting for between 16 and 66% of the energy within the stool.

This difference between gross and estimated metabolizable energy intakes should not be overlooked in studies of energy balance, particularly when attempts are made to compare individuals consuming diets of differing fibre content or with differing degrees of maldigestion or malabsorption, or both.

J.M. is a recipient of an MRC studentship. The support of Duphar Laboratories is gratefully acknowledged.

Merrill, A. L. & Watt, B. K. (1955). Energy value of foods, basis and derivation. *Agricultural Handbook* no. 74. Washington DC: US Department of Agriculture.

Paul, A. A. & Southgate, D. A. T. (1978). *McCance & Widdowson's The Composition of Foods.* London: H.M. Stationery Office.

Van de Kamer, J. H., Huinink, H. T. & Weijers, H. A. (1949). *Journal of Biological Chemistry* **177**, 347–355.

The use of antigliadin antibody assay in the assessment of dietary adherence in coeliac disease. By EVA DALY, C. P. KELLY, D. P. NUNES and M. J. GIBNEY, *Department of Clinical Medicine, Trinity College Dublin, Dublin 2, Republic of Ireland*

Antigliadin antibody assays are now used as a screening method for coeliac disease (CD). Less attention has been paid to their use in the follow-up of patients on a gluten-free diet (GFD). We studied serum IgG and IgA antigliadin antibody levels (Kelly *et al.* 1988) in seventy-one patients with biopsy-proven CD over a 3-year period. All patients had dietary assessments and a small-intestinal biopsy performed. Antigliadin antibody levels were also assessed in forty normal controls and in thirty-five patients with Crohn's disease. Results were analysed by the Mann-Whitney U test.

Serum IgA and IgG antibody assays showed good sensitivity and specificity in detecting untreated CD: IgA 87% and 91%, IgG 78% and 84% respectively. On serial measurements both assays were significantly lower in treated than in untreated patients ($P<0{\cdot}001$). IgA titres fell to a baseline level within 6 months of commencing a GFD, while the IgG antibody titres took up to 18 months to reach a baseline level. Patients (n 10) who did not adhere to a GFD showed no fall in antibody titres (Baker *et al.* 1975). Antigliadin antibody levels correlated better with dietary assessment (r 0·5) than with histological grading (r 0·32).

Six patients who had been on a GFD for a minimum of 12 months failed to show a histological response, despite demonstrating a fall in antigliadin antibody titres to low levels. No significant difference was seen in the dietary compliance of the responders and non-responders ($P=0{\cdot}66$). In assessing their failure to respond, serum zinc levels were measured in these six patients and in seven patients who responded to a GFD (Jones & Peters, 1981). Although the levels were significantly lower in the non-responders ($P=0{\cdot}008$) the mean values for both groups fell within the normal reference range of 10·7–18·1 μmol/l. However, two individual results from the non-responsive group were just below this normal range. Nonetheless in these non-responders low serum Zn would not appear to explain the absence of histological improvement on a GFD.

	No. of patients	Mean Zn (μmol/l)	Range
Non-responders	6	12·5	9·6–14·6
Responders	7	15·9	14·0–18·8

In conclusion the IgA antigliadin antibody assay was more sensitive and specific than the IgG assay in screening for untreated coeliac disease. Antigliadin antibody levels appear to reflect dietary adherence more closely than histological response. As the IgA antibody levels fall rapidly after introduction of a GFD, this assay appears to be a good method of confirming dietary adherence.

Baker, P. G., Barry, R. E. & Read, A. E. (1975). *British Medical Journal* **i**, 486–488.
Jones, P. & Peters, T. (1981). *Gut* **22**, 194–198.
Kelly, C. P., Feighery, C. F. & Weir, D. G. (1988). *Gastroenterology* **94**, A221.

Dietary assessment of iron intake as a reflection of Fe status. By M. COLLINS, *Dublin Institute of Technology, Kevin Street, Dublin* and E. MURPHY, H. SHORTT and D. GILL, *The Children's Hospital, Temple Street, Dublin, Republic of Ireland*

Most iron deficiency in children is of a dietary origin. It is also more prevalent in young children and more so in the underprivileged, with as many as 30% being anaemic and an additional 25% being Fe-deficient (Brault-Dubac *et al.* 1983; Ehrhardt, 1986). The Fe and nutritional status of twenty-two 1–5 year olds from Dublin was established using anthropometry and haematological indices of haemoglobin, serum Fe, transferrin, ferritin and total Fe binding capacity. This group was randomly selected by an independent third party. Nutrient intake was assessed by a 4-d dietary recall method, supported by a food frequency questionnaire.

	Mean	SD	Reference range
Total Fe intake (mg/d)	7·8	2·1	8–9
Available Fe (mg/d)	0·5	0·15	0·8–0·9
Availability of total Fe (%)	6·98	1·2	10
Haem intake (mg/d)	0·7	0·3	—
Non-haem intake (mg/d)	7·2	2	—
Absorption of non-haem Fe (%)	5·3	0·6	3–8
Haemoglobin (g/l)	115	8	115–165
Ferritin (μg/l)	18·6	20·9	10–330
Serum Fe (μmol/l)	11	4·4	10–30

It has been shown that total Fe intake and the availability of that Fe is a limiting factor in achieving adequate Fe status in this age group (Oski & Stockman, 1980). The availability of the Fe is dependent not only on the amount of Fe supplied in the diet but also on the nature of that Fe and the composition of the meal in which it is consumed (Monsen *et al.* 1978). In the present study total Fe intake and the availability of that Fe was calculated, taking into account enhancing and inhibiting factors in the diet (Monsen & Ballintfy, 1982). The results show that total Fe intake was low in this group and the percentage availability of that Fe was lower than the 10% assumed, being as low as 7%. This was reflected in the Fe stores of these toddlers as measured by serum ferritin, though it was not reflected in haemoglobin levels.

Brault-Dubac, M., Nadeau, M. & Dickie, J. (1983). *Human Nutrition: Applied Nutrition* **37A**, 210–221.
Ehrhardt, P. (1986). *British Medical Journal* **292**, 90–93.
Monsen, E. R. & Ballintfy, J. L. (1982). *Journal of the American Dietetic Association* **80**, 307–311.
Monsen, E. R., Halberg, L., Layrisse, M., Hegsted, D. M., Cook, J. D., Mertz, W. & Finch, C. A. (1978). *American Journal of Clinical Nutrition* **31**, 134–140.
Oski, F. A. & Stockman, J. A. (1980). *Pediatric Clinics of North America* **27**, 237–252.

Adipose tissue fatty acids in patients undergoing coronary artery angioplasty. By K. YOUNGER[1], B. FOLEY[2], P. CREAN[2], G. GEARTY[2], M. J. GIBNEY[1] and M. WALSH[2], [1]*Department of Clinical Medicine and Nutrition, Trinity College, Dublin* and [2]*Department of Cardiology, St James's Hospital, Dublin 8, Republic of Ireland*

Since 1977, percutaneous transluminal coronary angioplasty, in which a balloon-ended catheter is inflated inside the affected vessel(s) in order to enlarge the lumen, has increased dramatically as a therapeutic option in patients with ischaemic heart disease. However, restenosis is common, i.e. the vessel will become partially or completely blocked within 6 months in 30–50% of cases. The aim of the present study was to establish whether restenosis is related to the fatty acid composition of adipose tissue in angioplasty patients, as low dietary intakes of linoleic acid have been correlated with an increased risk of myocardial infarction (Wood *et al.* 1987). Since polyunsaturated fatty acids of the *n*–6 and *n*–3 series are not synthesized by the body in man, adipose tissue levels reflect long-term dietary intake of these fatty acids.

Eighty-two patients (sixty-two male, twenty female, mean age 52 (SD 9·1) years) who have undergone successful coronary angioplasty have been studied. They all had repeat coronary angiography at 6 months or sooner if they became symptomatic. Thirty-seven (45%) of the patients restenosed. Characteristics of patients are shown in Table 1; there were no significant differences between the patients who restenosed and those who did not. Subcutaneous fat biopsies were taken (in the sacro-iliac region) and analysed by gas–liquid chromatography using standard techniques. The major adipose tissue fatty acids (>14C chain length) are shown in Table 2.

Table 1. *Patient characteristics*

	Age (years)		No.of males	No. of females	Family history of coronary heart disease	Cigarette smoker			Total cholesterol (mmol/l)	
	Mean	SE				No	Yes	Ex	Mean	SE
Restenosed	54	1·4	31	6	17	16	8	13	6·1	0·19
Not restenosed	51	1·4	31	14	27	10	23	12	6·3	0·17

Table 2. *Adipose tissue fatty acids (% of total fatty acids) in patients undergoing angioplasty*

Fatty acid . . .		14:0	16:0	16:1	18:0	18:1	18:2	18:3	20:3	20:4	20:5	22:6
Restenosed	Mean	5·4	24·7	5·8***	4·1	41·0	12·8	1·5	0·2	0·5	0·2	0·2
(*n* 37)	SEM	0·37	0·48	0·38	0·25	0·54	0·78	0·08	0·03	0·03	0·04	0·02
Not restenosed	Mean	5·1	23·6	7·2	4·1	42·5	11·5	1·6	0·1	0·5	0·2	0·1
(*n* 45)	SEM	0·28	0·33	0·36	0·23	0·62	0·59	0·09	0·02	0·03	0·04	0·01

Significantly different from not restenosed (unpaired Student's *t* test): ***$P<0·001$.

There was no difference in linoleic (18:2 *n*–6) or arachidonic (20:4 *n*–6) acid levels between the patients who did not restenose and those who did. However, monounsaturated fatty acid levels were lower in the patients who restenosed. In the case of palmitoleic (16:1 *n*–7) acid the difference was highly significant ($P<0·001$), whereas with oleic (18:1 *n*–9) acid the difference just failed to reach statistical significance. These results are particularly interesting in view of the recent suggestion (Grundy, 1989) that monounsaturated fats (which may be endogenous or dietary in origin) may have a 'protective' role in coronary heart disease.

Grundy, S. M. (1989). *Journal of Nutrition* **119**, 529–533.

Wood, D. A., Riemersma, R. A., Butler, S., Thomson, M., MacIntyre, C., Elton, R. A. & Oliver, M. F. (1987). *Lancet* **i**, 177–183.

Vascularization in the epididymal and perirenal fat pads of young and adult mice. By J. M. KEARNEY and M. J. GIBNEY, *Division of Nutritional Sciences, Department of Clinical Medicine, Trinity College Medical School, St James's Hospital, Dublin 8, Republic of Ireland*

Adipose tissue triacylglycerols are believed to exist in two pools, one large pool with a slow turnover and one small pool with a rapid turnover. There is, however, very little known about the nature of these pools. They may exist in different adipose sites or within the same site and they may be structurally different with respect to triglyceride fatty acid composition. One possibility, which arose from work in our laboratory on the rate of uptake of labelled fatty acids by adipose tissue, is that vascularization determines metabolic activity. Little is known about the vascularization of white adipose tissue and the purpose of the present study was to examine vascularization both within and between mouse adipose tissue sites and at different ages.

The perirenal and epididymal fat pads were dissected out from ten young (3 weeks) and ten adult (3 months) male Laca mice. After embedding in wax, transverse and longitudinal sections (5 μm) were cut from the peripheral and core regions of each pad. The sections were stained using haemotoxylin and eosin, and the extent of vascularization measured using a light microscope linked to a Kontron image analyser. The results are presented in the Table for the log number of blood vessels per field of transverse sections. The data were analysed by analysis of variance.

	Young				Old			
	Perirenal		Epididymal		Perirenal		Epididymal	
	Mean	SE	Mean	SEM	Mean	SEM	Mean	SEM
Core	1·33	0·25	1·41	0·18	1·38	0·20	1·84	0·09
Peripheral	1·02	0·11	1·26	0·08	0·58	0·25	0·66	0·24

The number of blood vessels in a fat pad tended to be greatest in the core region ($P<0·05$) and this was most evident in young mice. In general there were little differences between the two fat pads. The effect of age and location within each fat pad was generally similar for transverse and longitudinal sections and also similar when the above results were expressed as blood vessel area rather than blood vessel number.

https://doi.org/10.1079/PNS19900014 Published online by Cambridge University Press

Histological changes in brown adipose tissue associated with weight loss in obese Zucker rats resulting from the combined administration of neuropeptide Y and noradrenaline. By A. AL-ARABI and J. F. ANDREWS, *Department of Physiology, Trinity College Dublin, Dublin 2, Republic of Ireland*

We have reported (Al-Arabi & Andrews, 1989) that combined treatment with neuropeptide Y (NPY) and noradrenaline (NA) caused significant weight loss in obese Zucker rats. Does this treatment have an effect on the function of brown adipose tissue (BAT) of these animals? We have studied the changes in the size of the triacylglycerol droplets (TGD) of BAT as an index of function (Suter, 1969) using two groups of adult male rats (twenty lean, Wistar strain; twenty obese (*fa/fa*), Zucker strain). Animals were acclimated to 28° (thermoneutrality). Each group was divided into five subgroups: (1) untreated controls, (2) carrier-treated controls, (3) NA-treated, (4) NPY-treated and (5) NPY+NA-treated. In subgroups 1–5 of each group, Alzet® (2002) osmotic minipumps were implanted under the skin in the interscapular region. Pumps were filled with carrier alone, 0·1 M-L-ascorbic acid and 0·02 M-L-Tiron® (Sigma Chemical Co, St Louis, Mo) (subgroup 2); carrier plus 0·3 M-NA (subgroup 3); carrier plus 0·3 μM-NPY (subgroup 4); or carrier plus both NA and NPY (subgroup 5). Delivery date of NPY was calculated to be 0·5 μg/h, and NA 20 μg/h extending over a period of 14 d. Sections of interscapular BAT samples obtained from randomly selected animals (one or more) from each group were examined and the cross-sectional areas of the TGD (*n* 500) were measured using the Kontron® image analysis system. Data were analysed using the Kolmogrov–Smirnov two-sample test.

TGD cross-sectional area (μm^2, median values)

Subgroup . . .	1	2	3	4	5
Genotype†	Untreated	Carrier	NA	NPY	NPY+NA
Obese	1673·0	1374·0	391·7**	1222·0*	18·2**
Lean	1174·0	356·5	184·4**	21·8**	13·4**

Significantly different from subgroup (2), carrier: *$P<0·05$, **$P<0·01$.
†Values are for 500 TGD per subgroup obtained from one or more animals.

The most significant finding was that the combined treatment had a dramatic effect in the obese where it caused the size of TGD to fall to values very similar to those of the NPY+NA-treated lean rats, indicating a thermogenic tissue. Together with the weight loss, these results suggest that the combined treatment with NPY and NA has a direct effect on the thermogenic activity of BAT and support our suggestion that NPY deficiency may be a factor related to the obesity of Zucker (*fa/fa*) rats.

Al-Arabi, A. & Andrews, J. F. (1989). *Proceedings of the Nutrition Society* **48**, 40A.
Suter, E. R. (1969). *Journal of Ultrastructure Research* **26**, 216–241.

At 30 min post-ingestion the thermic effect of feeding is increased by prior high-intensity exercise. By NIAMH BUCKLEY, MARY KELLY, B. DONNE and J. F. ANDREWS, *Department of Physiology, Trinity College Dublin, Dublin 2, Republic of Ireland*

Exercise can promote weight loss but the direct energy cost of the exercise accounts for only a fraction of the reduction in stores. It has been proposed that the balance is lost by exercise causing a sustained stimulation of resting metabolic rate. The findings are equivocal (review, Brehm, 1988); for example, Maehlum *et al.* (1986) reported a 16% elevation in resting metabolism for 12 h after exercise, whereas Goldberg *et al.* (1989) were unable to demonstrate any long-lasting increase in resting metabolic rate resulting from an exercise programme. Intensity of exercise is a variable which could possibly account for the different observations, alternatively exercise could decrease the efficiency of energy assimilation through increasing the thermic effect of feeding. We have investigated these two possibilities.

Sedentary, healthy young women (age range 20–27 years), having given informed consent, acted as subjects. They were studied on three separate occasions, each study being preceded by an overnight fast. Before the studies the onset of blood lactate accumulation (OBLA) was determined for each individual subject using a standard protocol involving stepwise increments of intensity of exercise on a bicycle ergometer, with blood lactate determined from a finger pin-prick at the end of each step. On two of the three test days, subjects were exercised either at a relatively low intensity (25 watts below their individual OBLA threshold for 1 h: mean pulse rate at end of exercise 162 (SEM 0·5) beats per min (bpm)), or at a relatively high intensity (25 watts above OBLA to exhaustion, mean pulse rate at exhaustion 185 (SEM 5) bpm, mean duration of exercise 13·3 (SEM 1·4) min). A standard meal was taken 120 min post-exercise (energy 3·15 MJ, g/kg: 650 carbohydrate, 250 protein, 100 fat). Oxygen consumption was determined by open-circuit indirect calorimetry (Douglas bag) with 5 min collections every 30 min. The study was done in a temperature-controlled room (21°). On a third day the study was repeated with no exercise preceding the test meal. Not every subject completed the full procedure. The order of study was randomized.

By 120 min post-exercise (before the meal) metabolic rate had fallen to near resting levels in both exercise groups: there was no significant sustained elevation and the two groups were indistinguishable. The meal caused a significant elevation in metabolism above immediate pre-meal values in all groups (150 min −120 min values, O_2 consumption (ml/kg per min), mean of individual differences): no prior exercise, +0·86 (SEM 0·20), n 5, $P<0·02$; prior low-intensity exercise, +1·18 (SEM 0·32), n 6, $P<0·002$; prior high-intensity exercise, +2·66 (SEM 0·22), n 4, $P<0·001$ (P determined by paired t test). Prior low-intensity long-duration exercise caused some increase in the thermic effect of feeding but this was not significant ($P<0·3$). However, high-intensity exercise to exhaustion caused a significant increase in the thermal effect of feeding with respect to both the no-exercising group ($P<0·001$) and the low-intensity exercise group ($P<0·025$) (unpaired t tests). We conclude that in general, prior exercise increases the energy cost of food assimilation, and that this effect is positively related to the intensity of exercise.

We confirm that exercise, even of high intensity, does not cause a sustained elevation of resting metabolic rate. Our findings may be a partial explanation of how exercise, by increasing the inefficiency of food storage mechanisms, causes weight loss.

Brehm, B. A. (1988). *Sports Medicine* **6**, 72–78.

Goldberg, G. R., Bingham, S. A., Coward, W. A. & Prentice, A. M. (1989). *British Journal of Nutrition* **48**, 155–173.

Maehlum, S., Grandmontagne, M., Newsholme, E. A. & Sejersted, O. M. (1986). *Metabolism* **35**, 425–429.

Increased retention of urea nitrogen in patients on total parenteral nutrition. By BRENDAN J. MORAN[1], S. J. KARRAN[1] and ALAN A. JACKSON[2], *Departments of* [1]*Surgery* and [2]*Human Nutrition, University of Southampton, Bassett Crescent East, Southampton SO9 3TU*

In the normal adult approximately 30% of the daily production of urea is retained by the body and hydrolysed in the lower bowel by the microflora. The nitrogen derived from urea hydrolysis may be used for protein synthesis, and the efficiency of utilization has been found to increase when the demand for N is in excess of that provided in the diet. This implies that the hydrolysis of urea can be an effective mechanism through which N is salvaged for the body.

The rate at which urea is hydrolysed may be used as a marker of microfloral activity, being significantly reduced by interventions that inhibit bacterial activity, such as antibiotics. It would appear that in the fasted state the metabolic activity of the colonic flora is decreased with a concomitant reduction in urea hydrolysis (Long *et al.* 1978). Patients on total parenteral nutrition (TPN) for intestinal failure have no oral intake and all nutrients are supplied by the intravenous route. The microflora are deprived of any exogenous source of substrate, and urea hydrolysis should be reduced to a minimum if the N supplied by the TPN is adequate.

In the present study the recovery and retention of urea N were measured in seven patients on TPN, two of whom were receiving broad-spectrum antibiotics, and compared with the findings in four healthy volunteers. A single dose of $^{15}N^{15}N$-urea was given intravenously (1·5 mg/kg) and urine was collected for 48 h. The recovery of ^{15}N was measured in urinary urea.

Recovery and retention of urea N in 48 h (expressed as % of the intravenous dose)

		Median recovery in urine		Retention	
	n	$^{15}N^{15}N$-urea	$^{15}N^{14}N$-urea	Median	Range
Normal controls	4	78	7	16	13–17
TPN	5	59	5	38	24–52
TPN on antibiotics	2	81	6	13	11–15

The retention of ^{15}N in the patients on TPN was significantly greater than that in the normal controls ($P<0·01$, Willcoxon rank sum test). Administration of antibiotics abolished this difference.

Contrary to expectation, there was significant hydrolysis of urea in the bowel of patients on TPN, and a substantial proportion of the N from urea was retained for further metabolic interaction. These results imply persistent activity of the colonic microflora, which was presumably supported by endogenous secretions. The extensive reutilization of the urea N suggests an imbalance between the demand for N and that supplied in the TPN solution.

Long, C. L., Jeevanandam, M. & Kinney, J. M. (1978). *American Journal of Clinical Nutrition* **31**, 1367–1382.

The intra-individual variation in urea kinetics measured in a single individual over a period of 4 years. By J. M. HIBBERT[1] and A. A. JACKSON[1,2], [1]*Faculty of Medical Sciences, University of the West Indies, Kingston 7, Jamaica* and [2]*Department of Human Nutrition, University of Southampton, Bassett Crescent East, Southampton SO9 3TU*

Nitrogen balance can be maintained over a wide range of dietary intakes, by modifying the rate at which urea is produced and excreted. In normal adults on an adequate diet about 30% of urea production is retained in the body, with the N being made available for metabolic interaction following hydrolysis by the microflora of the lower bowel. Different groups have adopted different approaches to the measurement of urea kinetics, but all have noted a wide range of intra-individual variation in the results obtained. It is not clear to what extent this variation may be accounted for by methodological differences or the conditions under which the studies have been performed. In the present study a standard approach was adopted for the measurement of urea kinetics on five occasions in a single adult female over a period of 4 years.

No attempt was made to control the previous intake or level of activity and the studies were carried out on days 2, 6, 20 and 24 of the menstrual cycle. Urea kinetics were measured using a prime/intermittent oral dose of $^{15}N^{15}N$-urea at 0, 6, 9, 12 and 15 h. A standard intake of food was given, and urine was collected at each of the time intervals up to 18 h. Urea was isolated from urine and the excretion of $^{15}N^{15}N$-urea and $^{15}N^{14}N$-urea measured. Urea kinetics were calculated as described previously (Jackson *et al.* 1984).

Plateau labelling of urinary urea was achieved in all the studies.

Study	Protein intake (mg N/kg per d)	Urea production (mg N/kg per d)	Urinary urea excretion (mg N/kg per d)	Bowel urea hydrolysis (mg N/kg per d)
1	228	201	156	45
2	193	167	107	60
3	255	193	137	56
4	232	229	175	54
5	247	201	140	60
Mean	231	198	143	55
Coefficient of variation (%)	10	11	18	11

Urea production was 86% of N intake and 72% of production was excreted in the urine with 28% being hydrolysed and available for further metabolism. Small differences in the intake could account for some of the variation in urea kinetics, with the coefficient of variation for urea production and hydrolysis being no greater than that for N intake. The highest variation was seen in urea excretion: in this study no attempt was made to correct for changes in urea pool size.

The results show that intra-individual variations in urea kinetics may be substantially less than the inter-individual variations reported in the literature. For example, Walser & Bodenlos (1959) found coefficients of variation of 72% for production, 32% for excretion and 75% for hydrolysis in normal adults.

Jackson, A. A., Picou, D. & Landman, J. (1984). *Human Nutrition: Clinical Nutrition* **38C**, 339–354.
Walser, M. & Bodenlos, L. J. (1959). *Journal of Clinical Investigation* **38**, 1617–1626.

The effect of copper deficiency on plasma homocysteine in the rat. By J. C. W. BROWN, G. MAZDAI and J. J. STRAIN, *Biomedical Sciences Research Centre, University of Ulster at Jordanstown, Newtownabbey, Co. Antrim BT37 0QB* and A. HEWITT, *Veterinary Research Laboratories, Belfast BT4 3SD, Northern Ireland*

Excessive levels of dietary methionine (Lynch & Strain, 1989) or its transulphuration metabolite, homocysteine (Brown & Strain, 1989), can markedly decrease copper status in the rat. The possibility that Cu deficiency might exert an effect on methionine metabolism, and thereby increase homocysteine levels, was investigated in the present study.

Three groups (*n* 8) of male weanling Sprague-Dawley rats were provided with deionized water and given the experimental diets for 8 weeks. Group DF was fed on a Cu-deficient diet (1·02 mg/kg diet) *ad lib.*, control group PF received a diet adequate in Cu (14·83 mg/kg diet) and was pair-fed with the DF group, and control group AL consumed the diet adequate in Cu *ad lib*. Hepatic levels of Cu and the activities of the Cu-dependent enzymes, cytochrome *c* oxidase (CCO; *EC* 1.9.3.1) and superoxide dismutase (Cu/Zn-SOD; *EC* 1.15.1.1), together with plasma caeruloplasmin (CP; *EC* 1.16.3.1), were used as measures of Cu status.

Group . . .	AL		PF		DF	
	Mean	SEM	Mean	SEM	Mean	SEM
Body-wt (g)	404[a]	17·5	317[b]	13·2	259[c]	12·9
Plasma CP (U/l)	189·5	16·56	176·2	10·55	ND	
Plasma vitamin B_{12} (ng/l)	1441[a]	165·4	1370[a]	141·3	1804[a]	217·7
Plasma folate (μg/l)	86·5[a]	2·58	77·5[b]	3·06	77·3[b]	7·32
Plasma homocysteine (μmol/l)	17·27[a]	0·68	12·70[b]	0·63	15·56[ab]	1·73
Erythrocyte folate (μg/g protein)	4·17[ab]	0·52	2·95[a]	0·23	4·76[b]	0·76
Liver Cu (μg/g dry wt)	11·44[a]	0·55	14·15[a]	2·09	2·03[b]	0·29
Liver CCO (U/mg protein)	6·43[a]	1·05	3·44[b]	1·08	0·80[c]	0·23
Liver Cu/Zn-SOD (U/mg protein)	123·5[a]	17·70	112·9[a]	20·04	35·0[b]	6·94

ND, not detectable.

[a–c] Means in horizontal rows with different superscripts are significantly different by the least significant difference (LSD) test: $P<0·05$.

All measurements of Cu status were greatly decreased in the DF group. Plasma homocysteine in the PF group was significantly lower than that in the AL group but was not significantly different from the DF group. Although there was no significant increase in plasma homocysteine with Cu deficiency, a possible effect of Cu deficiency on one of the co-factors in methionine/homocysteine metabolism was suggested by the significant increase in erythrocyte folate in the DF group compared with the PF group.

Brown, J. C. W. & Strain, J. J. (1989). *Proceedings of the Nutrition Society* **48**, 160A.
Lynch, S. M. & Strain, J. J. (1989). *Free Radical Research Communications* **5**, 221–226.

No diminution of complement activity or oxidative respiratory burst capacity with copper deficiency in the rat immune system. By G. MAZDAI, J. C. W. BROWN, B. M. HANNIGAN and J. J. STRAIN, *Biomedical Sciences Research Centre, University of Ulster, Coleraine BT52 1SA, Northern Ireland*

Copper deficiency has been shown to affect the host defence system of certain experimental animals. In Cu-deficient conditions cells of lymphoid origin have decreased responses, including lowered antibody production, whilst phagocytic cells have decreased microbiocidal activity (Koller *et al.* 1987).

In the present study the effect of Cu-deficiency on humoral and cellular components of the immune system of male weanling Sprague-Dawley rats were investigated. Three groups (*n* 8) of rats were provided with deionized water and fed on the experimental diets for 8 weeks. Group DF was fed on a Cu-deficient diet *ad lib.*, control group PF received a diet adequate in Cu and was pair-fed with the DF group, and control group AL consumed the diet adequate in Cu *ad lib*. After 8 weeks neutrophils were isolated from fresh heparinized blood, and upon phorbol myristate acetate (PMA) stimulation, oxidative respiratory burst (ORB) capacity was evaluated by cytochrome *c* reduction (Leslie & Allen, 1987). Serum was also obtained and stored at −70° before analysis for complement activity using the method of Kabat & Mayer (1971). The DF group was deemed to be Cu-deficient by biochemical criteria.

Group . . .	AL		PF		DF	
	Mean	SEM	Mean	SEM	Mean	SEM
Complement activity (C'H:50 Units/ml)†	48·5	23·0	46·8	32·2	48·0	13·0
Granulocyte ORB capacity (O_2^- nM/10^5 cells)	3·1	1·1	4·3	5·4	13·2	18·8
Thymus wt (g/kg body-wt)	2·18	0·4	2·34	0·5	1·86**	0·4

†C'H:50 Units/ml is the amount of complement activity in 1 ml of undiluted serum necessary to bring about 50% lysis of optimally sensitized sheep erythrocytes.

Significantly different from PF (*t* test): $^{**}P<0{\cdot}01$.

Although individual variations in these indices were high, results suggest that there was no diminution of complement activity or ORB capacity with Cu-deficiency. Thymus weight, however, was significantly decreased in DF relative to PF animals. This may underlie some of the findings of other groups who showed decreased T-cell numbers and responsiveness in Cu-deficiency. Therefore these findings support Koller *et al.* (1987) who proposed that Cu-deficiency does not result in a wide spectrum of changes in the immune system. It seems that only certain immunological components are Cu-dependent.

Kabat, E. A. & Mayer, M. M. (1971). *Experimental Immunochemistry*, 2nd ed, pp. 135–153. Springfield, IL: Charles C. Thomas.

Koller, L. D., Mulhern, S. A., Frankel, N. C., Steven, M. G. & Williams, J. R. (1987). *American Journal of Clinical Nutrition* **45**, 997–1006.

Leslie, R. G. Q. & Allen, R. (1987). *Journal of Immunological Methods* **103**, 261–266.

Absorption of iron and zinc from soya- and cow's milk-based infant formulae in sucking rats. By M. M. BRENNAN, A. FLYNN and P. A. MORRISSEY, *Department of Nutrition, University College, Cork, Republic of Ireland*

Soya-based infant formulae are used as alternatives to cow's milk-based formulae for infants with disorders such as lactose intolerance and cow's milk protein allergy. Since there is considerable evidence that the bioavailability of minerals and trace elements in soya-bean products is low, the present study was carried out to investigate the bioavailability of iron and zinc in soya- and cow's milk-based formulae. The method used was developed for simultaneous studies of Fe and Zn absorption in sucking rats (Brennan *et al.* 1989).

Two soya-based infant formulae (Wysoy, Wyeth Laboratories and Formula S, Cow and Gate Ltd) and one cow's milk-based formula (SMA White Cap, Wyeth Laboratories) were reconstituted, extrinsically labelled with both ^{59}Fe (1 μCi/ml) and ^{65}Zn (μCi/ml) and 0·6 ml was given by gavage to 17-d-old rats, previously fasted for 18 h. Animals were killed 6 h later and stomach, small intestine (SI), caecum–colon and liver removed. Small intestines were perfused with 6 ml saline (0·15 M-sodium chloride). ^{59}Fe and ^{65}Zn in tissues were determined in a well gamma counter using a channels ratio method.

Absorption and transfer to carcass of both ^{59}Fe and ^{65}Zn were lower from the soya-based formulae than from the cow's milk-based formula. This difference was much more marked for ^{65}Zn than for ^{59}Fe and a high proportion of the ^{65}Zn in both soya-based formulae was present in unabsorbable form. Absorption and transfer of ^{65}Zn were lower from Formula S than from Wysoy.

Uptake (% dose) of ^{59}Fe and ^{65}Zn from soya- and cow's milk-based infant formulae in sucking rats

	SMA White Cap (*n* 6)		Wysoy (*n* 6)		Formula S (*n* 6)	
Organ/tissue	Mean	SEM	Mean	SEM	Mean	SEM
^{59}Fe						
Stomach	0·8	0·1	0·6	0·1	0·8	0·1
SI	7·6	0·6	5·7	0·3	5·4*	0·5
SI perfusate	0·4	0·2	0·6	0·2	0·5	0·2
Caecum–colon	4·2	1·5	19·9*	2·2	17·8*	0·9
Liver	12·4	0·9	11·6	0·5	11·3	0·5
Absorbed†	94·5	1·5	79·6*	2·4	81·0*	0·8
Carcass‡	86·9	2·1	72·8*	1·8	75·5*	0·8
^{65}Zn						
Stomach	1·9	0·2	0·9	0·1	1·1	0·2
SI	22·5	2·2	13·4**	0·9	9·8**	1·1
SI perfusate	3·6	0·4	3·5	1·4	2·6	0·8
Caecum–colon	9·2	2·5	51·4**	5·1	67·6**	3·3
Liver	23·5	0·7	13·5*	1·7	8·6**	0·9
Absorbed†	85·3	2·5	43·3**	4·5	28·7**	2·8
Carcass‡	62·9	1·4	30·9**	4·6	18·8**	2·4

Significantly different from SMA White Cap: *$P<0·05$, **$P<0·01$.
†Absorbed = 100 − (stomach + SI perfusate + caecum–colon) (%).
‡Carcass = absorbed − SI.

These results indicate that the bioavailability of Fe and Zn in soya-based infant formulae is lower than that in cow's milk-based formulae. This should be considered when establishing fortification levels of Fe and especially Zn for soya-based formulae.

Brennan, M., Flynn, A. & Morrissey, P. A. (1989). *Proceedings of the Nutrition Society* **48**, 39A.

Total peroxyl radical-trapping antioxidant potential of serum in myocardial infarction.

By C. W. MULHOLLAND, *Biochemistry Department, Whiteabbey Hospital, Belfast* and J. J. STRAIN, *Biomedical Sciences Research Centre, University of Ulster at Jordanstown, Newtownabbey, Co. Antrim BT37 0QB, Northern Ireland*

Recent evidence suggests that the extent of injury sustained by the heart after an ischaemic insult is in part related to damage caused by free radicals, particularly at the time of reperfusion (McCord & Roy, 1982). Antioxidant nutrients are involved in the body's complex defence mechanism against free radical injury and mounting evidence suggests that antioxidants do not function in isolation but act synergistically to produce an overall effect greater than the sum of the individual components (Gey, 1986). The total peroxyl radical-trapping antioxidant potential (TRAP) assay, as developed by Wayner *et al.* (1987), is a specific measure of the capacity of the secondary antioxidants, α-tocopherol, ascorbic acid, reduced thiol compounds and urate, in serum to resist controlled in vitro peroxidation.

Experimental TRAP values from fifteen patients who had suffered an acute myocardial infarction (based on clinical, electrocardiographic and biochemical criteria) were compared with fifteen age- and sex-matched controls (Table).

		Experimental TRAP (μmol peroxyl radical trapped/l of serum)	
	n	Median	Range
Patients	15	672**	418– 899
Controls	15	867	648–1080

Mann-Whitney test: $^{**}P<0{\cdot}01$.

Ascorbate (reduced) levels were also significantly lower ($P<0{\cdot}05$, Mann-Whitney) in patients (median, 23·9 μmol/l; range, 14·8–56·8) compared with the controls (median, 50·2 μmol/l; range, 19·9–95·5) but the concentrations of reduced thiol compounds, α-tocopherol and urate in the two groups were not significantly different.

These preliminary results demonstrate that the combined potential of the known secondary antioxidants to protect against a peroxyl radical challenge in vitro is decreased in serum from myocardial infarction patients. However, the limitations inherent in this type of study make it difficult to form any conclusions on cause or effect.

Gey, K. F. (1986). *Bibliotheca Nutritio et Dieta* **37**, 53–94.
McCord, J. M. & Roy, R. S. (1982). *Canadian Journal of Physiology and Pharmacology* **60**, 1346–1352.
Wayner, D. D. M., Burton, G. W., Ingold, K. U., Barclay, L. R. C. & Locke, S. J. (1987). *Biochimica et Biophysica Acta* **924**, 408–419.

Bacterial fermentation occurs in the terminal ileum of ileostomates. By J. DOWSETT, M. J. GIBNEY and N. P. KENNEDY, *Division of Nutritional Sciences, Department of Clinical Medicine, Trinity College Medical School, St James's Hospital, Dublin 8, Republic of Ireland*

Ileostomates have frequently been used as controls in carbohydrate absorption studies in man, presuming them to lack a significant bacterial flora (Chapman *et al.* 1985). However, substantial bacterial colonization of the terminal ileum in ileostomates has been reported (Gorbach *et al.* 1967). If this leads to significant fermentation in this group, their validity as controls is lessened.

We studied bacterial fermentation in ten ileostomates following each of three carbohydrate meals of differing composition (50 g glucose, 25 g lactulose, or 170 g rice providing 100 g carbohydrate). To measure fermentation, the hydrogen breath test (indirect method) and estimation of volatile fatty acids (VFA) (direct method) in faecal effluent were performed. Fasting breath hydrogen was assessed in ten normal volunteers for comparison. Fasting levels of breath hydrogen (ppm) were significantly higher in the control group than in the ileostomates (mean 9·6 (SD 3·8), mean 2·2 (SD 1·6) respectively, $P<0·05$). Results of the VFA analyses are shown in the Table.

Volatile fatty acid concentration (mmol/l)

Meal		Acetic	Proprionic	Butyric
Glucose	Mean	22·9	1·8	3·2
	SEM	6·4	0·5	1·5
Lactulose	Mean	9·7	0·12	0·62
	SEM	3·6	0·07	0·23
Rice	Mean	43·5	2·17	5·3
	SEM	8·9	0·68	2·1

The proportions of faecal VFA compare favourably with those reported for normal populations (Weaver *et al.* 1989). Their presence confirms the existence of bacterial fermentation in the ileum of ileostomates, which must be considered in the interpretation of carbohydrate absorption studies where patients with ileostomies are used as controls. Lower fasting breath hydrogen values in ileostomates, probably indicating a lesser intestinal bacterial mass, did not vary following different test meals. This implies that this test is insensitive in detecting differences in fermentation in those patients. The degree of osmotic dilution occurring after ingestion of lactulose by these patients limits the value of lactulose as a test meal in ileostomates.

Chapman, R. W., Sillery, J. K., Graham, M. M. & Saunders, D. R. (1985). *American Journal of Clinical Nutrition* **41,** 1244–1248.

Gorbach, S. C., Nahas, L., Weinstein, L., Levitan, R. & Patterson, J. F. (1967). *Gastroenterology* **53,** 874–880.

Weaver, J. A., Krause, G. A., Miller, T. L. & Wolin, N. J. (1989). *Gut* **30,** 19–25.

Effect of α-tocopherol supplementation of sow's diet on milk α-tocopherol levels and on α-tocopherol transfer to the suckling piglet. By F. J. MONAHAN, P. A. MORRISSEY and D. J. BUCKLEY, *National Food Biotechnology Centre, University College, Cork, Republic of Ireland* and J. I. GRAY *Department of Food Science and Human Nutrition, Michigan State University East Lansing, MI 48824, USA* and P. B. LYNCH *Teagasc, Moorepark, Fermoy, Co. Cork, Republic of Ireland*

Vitamin E (α-tocopherol) functions as an antioxidant and prevents free radical-initiated lipid peroxidation in animal tissues (Machlin, 1984). The young piglet is vulnerable to vitamin E deficiency, particularly following parenteral iron administration (Tollerz, 1973). In the present study the effect of the α-tocopherol content of the sow's diet on α-tocopherol uptake by the suckling piglet was investigated.

Twelve sows were randomly divided into two groups of six at day 3 postpartum. One group was fed on a control diet containing 30 mg α-tocopherol acetate/kg feed while the other group was fed on an α-tocopherol-enriched diet containing 200 mg α-tocopherol acetate/kg feed. Blood and milk samples were taken from both groups of sows throughout the nursing period and blood samples were taken from their respective piglets at weaning. α-Tocopherol levels in milk and plasma were measured by high performance liquid chromatography.

Effect of α-tocopherol supplementation of sow's feed on α-tocopherol content of milk, sow plasma and piglet plasma

α-tocopherol in:	Days after supplementation	Control (*n* 6) Mean	Control (*n* 6) SEM	Supplemented (*n* 6) Mean	Supplemented (*n* 6) SEM
Sow plasma (μg/ml)	0	1·9	0·24	2·2	0·12
	21	2·7	0·37	4·0*	0·32
Milk (μg/g fat)	0	65·4	9·32	65·5	5·67
	3	51·4	6·49	97·5**	12·8
	7	54·7	8·91	91·5*	9·02
	14	55·1	5·17	98·5**	10·8
	21	59·4	7·76	103·6*	12·2
Piglet plasma (μg/ml)	21	3·1	0·14	4·5*	0·49

Significantly different from control (unpaired *t* test): $*P<0·05$, $**P<0·01$.

Sows fed on the α-tocopherol-enriched diet had significantly higher levels of milk α-tocopherol (μg/g fat) at day 3 of supplementation. The differences between the two groups were maintained over the 3-week sampling period. At weaning (day 21), piglets receiving milk from sows fed on the α-tocopherol-enriched diet had significantly higher plasma levels of α-tocopherol.

These results show that α-tocopherol can be efficiently transferred from the sow's diet, via the milk, to the suckling piglet, thereby improving the biological antioxidant status of the piglet.

Machlin, L. J. (1984). In *Handbook of Vitamins. Nutritional, Biochemical and Clinical Aspects*, pp. 99–145 [L. J. Machlin, editor]. New York: Marcel Dekker Inc.

Tollerz, G. (1973). *Acta Agriculturae Scandinavica*, Suppl. 19, 184–187.

The effect of heated sunflower oil on concentrations of α-tocopherol and thiobarbituric acid-reacting substances in chick tissues. By P. J. A. SHEEHY, P. A. MORRISSEY and A. FLYNN, *Department of Nutrition, University College, Cork, Republic of Ireland*

Consumption of thermally oxidized rapeseed oil by rats is associated with reduced hepatic and serum tocopherol status, and increased levels of thiobarbituric acid-reacting substances (TBARS) in liver (Izaki *et al.* 1984). Heated polyunsaturated oils also exhibit considerable toxicity when fed to chicks (Budowski *et al.* 1979). The objective of the present study was to investigate the effects of heated sunflower oil on the concentrations of α-tocopherol and TBARS numbers in a variety of chicken tissues.

Thirty-six ISA Brown chicks were divided into three groups and were fed on diets containing 40 g sunflower oil/kg which was fresh (FO), or heated (120° for 11 h) (HO) or heated and supplemented with α-tocopherol acetate (HE). Concentrations of α-tocopherol in diets were 30 μg/g (FO), 25 μg/g (HE) and 1·3 μg/g (HO). After 32 d, chicks were killed by cervical dislocation, plasma and tissues were removed and stored at −20° until required.

The concentrations of α-tocopherol and TBARS numbers in various tissues are shown in the Table.

	α-Tocopherol†							TBARS‡					
	FO		HO		HE		HE:FO	FO		HO		HE	
Organ/Tissue	Mean	SE	Mean	SE	Mean	SE	Mean	Mean	SE	Mean	SE	Mean	SE
Plasma	16·6[a]	1·40	0·83[c]	0·08	6·65[b]	1·01	0·40	5·38[b]	0·28	15·0[a]	1·27	5·90[b]	0·41
Liver	135[a]	25·9	3·00[c]	0·96	40·0[b]	2·20	0·30	0·49[a]	0·10	0·36[a]	0·08	0·36[a]	0·07
Heart	326[a]	54·1	10·5[c]	1·80	160[b]	5·50	0·33	0·68[a]	0·01	0·79[a]	0·05	0·74[a]	0·05
Lung	147[a]	32·2	14·0[c]	6·20	64·6[b]	8·80	0·44	1·29[b]	0·04	1·55[a]	0·09	1·59[a]	0·08
Spleen	121[a]	10·3	4·53[c]	0·65	61·0[b]	4·10	0·50	1·24[b]	0·07	1·55[a]	0·06	1·33[ab]	0·08
Thigh muscle	58·1[a]	4·30	6·07[c]	0·79	33·4[b]	4·30	0·57	1·23[a]	0·13	1·34[a]	0·11	1·29[a]	0·06
Breast muscle	28·8[a]	1·80	4·59[c]	0·44	19·1[b]	2·10	0·67	0·70[b]	0·07	0·92[a]	0·07	0·82[ab]	0·03
Cerebellum	24·3[a]	1·60	12·9[b]	2·60	16·5[b]	2·80	0·69	3·80[b]	0·27	4·79[a]	0·33	3·66[ab]	0·41
Cerebrum	25·1[a]	2·80	11·8[b]	1·50	18·7[a]	1·36	0·76	3·84[b]	0·14	3·37[a]	0·19	3·45[a]	0·43

[a–c] Mean values in horizontal rows not sharing a common superscript letter are significantly different (unpaired *t* test): $P<0·05$.

†μg/ml plasma or ng/mg tissue protein.

‡nmol malondialdehyde/ml plasma or nmol malondialdehyde/mg tissue protein.

With the exception of HE cerebrum, the α-tocopherol status of HO and HE tissues was significantly reduced relative to FO tissues. The depression in HE α-tocopherol compared with FO values (HE:FO) was more pronounced in tocopherol-rich tissues. TBARS numbers in HE lung and in HO breast muscle, lung, spleen, cerebellum and plasma, were significantly increased compared with those of the FO group.

The results suggest that ingestion of heated sunflower oil induces in vivo lipid peroxidation in several tissues. Reductions in the rate of peroxidation were observed in various tissues from chicks fed on heated oil supplemented with α-tocopherol.

Budowski, P., Bartov, I., Dror, Y. & Frankel, E. N. (1979). *Lipids* **14**, 768–772.
Izaki, Y., Yoshikawa, S. & Uchiyama, M. (1984). *Lipids* **19**, 324–331.

Effect of dietary fish oil on rat lymphocyte proliferation in vitro. By D. M. CANTILLON[1], P. A. MORRISSEY[1], T. P. O'CONNOR[2] and A. FLYNN[1], [1]*Department of Nutrition* and [2]*Department of Food Chemistry, University College, Cork, Republic of Ireland*

High *n*-3 polyunsaturated fatty acid intake is known to alter phospholipid profiles and decrease prostaglandin synthesis. Since prostaglandin E_2 has been shown to suppress the mitogenic response of T-cells (Rogers *et al.* 1980), it is likely that high *n*-3 intake may play a significant role in immune function.

Two experiments were conducted. In each experiment, three groups of rats were fed on diets containing 50 g olive oil, 50 g maize oil or 50 g cod liver oil/kg for 3 weeks. Olive oil was used as a control and maize and cod liver oils as good sources of *n*-6 and *n*-3 fatty acids respectively. In Expt 1, 50 mg vitamin E/kg diet was used. In Expt 2, 150 mg vitamin E/kg diet was used. The higher level of vitamin E was added to minimize in vivo lipid peroxidation.

Rats fed on the *n*-3-enriched diet showed a significant decrease in arachidonic acid content (20:4 *n*-6) of spleen phospholipids, but had significantly higher levels of eicosapentaenoic (20:5 *n*-3) and docosahexaenoic acids (22:6 *n*-3) in both experiments. The changes in spleen phospholipid, however, were not as pronounced in rats fed on the lower level of vitamin E.

Effect of dietary fat on fatty acid composition of rat spleen phospholipids (wt % total fatty acids)

(Mean values with their standard errors for six samples per group)

	Expt 1						Expt 2					
Fatty acid	Cod liver oil		Maize oil		Olive oil		Cod liver oil		Maize oil		Olive oil	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Linoleic	11·03	0·6	7·44	0·5	12·10	1·1	5·50	0·4	12·66	0·9	11·48	0·1
Linolenic	1·65	0·3	0·64	0·2	0·94	0·1	3·10	0·1	0·92	0·4	1·20	0·1
Arachidonic	9·31	0·7	18·16	1·0	18·14	0·1	7·30	0·6	19·22	1·4	18·80	0·1
Eicosapentaenoic	4·30	0·3	ND		0·44	0·1	5·40	0·5	ND		0·68	0·1
Docosahexaenoic	4·80	0·2	0·98	0·1	0·84	0·2	5·51	0·4	1·30	0·1	1·18	0·1
Saturates	47		48		47		42		47		44	
Monounsaturates	22		25		20		24		19		27	
Polyunsaturates	31		27		33		34		34		29	
n-6:*n*-3 ratio	1·9		15·8		13·6		0·91		14·3		9·9	

ND, not detected.

Splenocytes from each group were cultured in fetal bovine serum and examined for mitogen-induced (concanavalin A) blastogenesis. No significant difference in mitogen-induced [^{3}H]thymidine uptake was observed when 50 mg vitamin E was used in the diet. However, when higher vitamin E levels (150 mg/kg of diet) were given, significantly higher [^{3}H]thymidine uptake was observed in the *n*-3 fatty acid-enriched splenocytes.

Rogers, T. J., Nowowiejski, I. & Webb, D. R. (1980). *Cellular Immunology* **50**, 82–89.

The effect of a fish oil supplement on the phospholipid fatty acid composition of plasma, platelets, neutrophils, lymphocytes and monocytes. By B. HUNTER and M. J. GIBNEY, *Division of Nutritional Sciences, Department of Clinical Medicine, Trinity College Dublin Medical School, St James's Hospital, Dublin 8, Republic of Ireland*

Marine oils, rich in the *n*-3 polyunsaturated fatty acid, eicosapentaenoic acid (EPA), give rise to the 3-series prostaglandins and leukotrienes with reduced biological potency. Many animal studies and cell culture studies have been carried out and have demonstrated an immunosuppressive effect of fish oils. These have been complemented by a limited number of human studies. In the animal studies, the fish oil is often given at high doses and during a period of growth which may overestimate the potential to modify human blood cells at the normal hypolipidaemic dose of 10 g fish oil/d.

The purpose of the present study was to examine the uptake of EPA into blood cells of healthy volunteers (*n* 7) consuming a fish oil (MaxEpa) supplement of 15 g/d. Blood samples were taken at day 0, and at 2 and 12 weeks supplementation. The results are summarized in the Table for EPA (% w/w of phospholipid fatty acids):

Week . . .	0		2		12	
	Mean	SD	Mean	SD	Mean	SD
Plasma	0·9	0·2	4·3*	1·6	3·4*	1·5
Platelet	0·7	0·1	2·6*	0·5	2·4*	1·2
Neutrophils	1·2	0·8	1·7*	0·2	2·7*	0·8
T-lymphocytes	1·1	0·3	2·9*	0·5	3·2*	0·9
B-lymphocytes	1·2	0·2	3·4*	0·5	3·1*	0·6
Monocytes	1·0	0·3	2·2*	0·4	2·8*	0·9

*Significantly different from control (analysis of variance): *$P<0·05$.

EPA was rapidly incorporated into all cells, including those with short half-lives such as neutrophils and those with long half-lives such as lymphocytes. Another *n*-3 long-chain fatty acid present in fish oil, docosahexaenoic acid, increased significantly in plasma but did not increase in any of the cells.

In general, the uptake of EPA was accompanied by a fall in the level of phospholipid arachidonic acid which was significant ($P<0·05$) in the case of plasma and T-lymphocytes at both 2 and 12 weeks. In the case of B-lymphocytes, neutrophils and platelets, the decline in arachidonic acid only became significant at week 12.